

(FILE 'HOME' ENTERED AT 12:56:08 ON 16 JAN 2003)

FILE 'CAPLUS, MEDLINE' ENTERED AT 12:56:19 ON 16 JAN 2003

FILE 'MEDLINE, CAPLUS' ENTERED AT 12:56:26 ON 16 JAN 2003

L1 1966 S NICOTINE AND (HEARTBEAT OR BLOOD PRESSURE)
L2 37 S NICOTINE (W) (HEARTBEAT OR BLOOD PRESSURE)
L3 25 S L2 AND (INCREASE? OR ELEVATED)
L4 0 S (NICOTENE) (W) (SMOK?) (W) (EPINEPHRINE OR NOREPINEPHRINE)
L5 0 S (NICOTENE) AND (SMOK?) AND (EPINEPHRINE OR NOREPINEPHRINE)
L6 0 S (NICOTENE) AND (SMOK?) AND (EPINEPHRINE OR NOREPINEPHRINE)
L7 215 S (NICOTINE) AND (SMOK?) AND (EPINEPHRINE OR NOREPINEPHRINE)
L8 19 S L7 AND STRESS?

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L3 ANSWER 1 OF 25 MEDLINE
 AN 1998147543 MEDLINE
 DN 98147543 PubMed ID: 9488219
 TI Effects of cigarette smoking on carotid and radial artery distensibility.
 AU Failla M; Grappiolo A; Carugo S; Calchera I; Giannattasio C; Mancina G
 CS Cattedra di Medicina Interna I, Universita degli Studi di Milano and
 Ospedale S. Gerardo di Monza, Milan, Italy.
 SO JOURNAL OF HYPERTENSION, (1997 Dec) 15 (12 Pt 2) 1659-64.
 Journal code: 8306882. ISSN: 0263-6352.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199804
 ED Entered STN: 19980410
 Last Updated on STN: 19980410
 Entered Medline: 19980402
 AB OBJECTIVE: Cigarette smoking acutely induces a marked **increase**
 of blood pressure and heart rate. This is accompanied by a marked
 reduction of radial artery distensibility. Whether this reflects an
 alteration of arterial mechanics of large elastic arteries as well is not
 established, however. DESIGN AND METHODS: In this study we addressed the
 acute effects of smoking on the stiffness of a peripheral medium-sized
 muscular artery and a large elastic vessel. We studied seven healthy
 normotensive smokers (age 28+/-7 years, mean+/-SEM), in the absence of
 smoking for at least 24 h. Radial artery (NIUS 02) and carotid artery
 diameter (WTS) were concomitantly acquired beat-to-beat in the 5 min
 before, during and after smoking of a cigarette containing 1.2 mg of
nicotine. **Blood pressure** and heart rate were
 concomitantly recorded by a Finapres device. Radial and carotid artery
 distensibility were calculated according to the Langewouters and Reneman
 formulae, respectively. Data were collected for consecutive 30 s periods.
 Statistical comparisons were performed between the three different phases
 and, within each phase, between 30 s periods. In five subjects the
 protocol was repeated after 1 week using a straw rather than a cigarette
 to obtain data under sham smoking. RESULTS: Smoking **increased**
 systolic blood pressure by 14%, diastolic blood pressure by 10% and heart
 rate by 27%. Radial artery diameter was reduced during smoking (-3.7%) and
 more so after smoking (-14.8%), while carotid artery diameter did not
 change significantly either during or after smoking. Radial artery
 distensibility was also significantly reduced only after smoking (-41.3%,
 P < 0.01), while carotid artery distensibility was significantly reduced
 both during (-33.3%) and after smoking (-27.2%) (P < 0.01 versus before).
 No changes in blood pressure, heart rate and arterial wall mechanics were
 induced by sham smoking. CONCLUSIONS: Acute cigarette smoking reduces
 distensibility not only in medium-sized but also in large elastic
 arteries, therefore causing a systemic artery stiffening. The mechanisms
 of these effects remain to be determined. However, it is likely that
 adrenergic mechanisms are responsible for the arterial distensibility
 alterations.

L3 ANSWER 3 OF 25 CAPLUS COPYRIGHT 2003 ACS

AN 1990:454204 CAPLUS

DN 113:54204

TI Tolerance to nicotine-induced sympathoadrenal stimulation and cross-tolerance to stress: differential central and peripheral mechanisms in rats

AU Kiritsy-Roy, Judith A.; Mousa, S. A.; Appel, N. M.; Van Loon, G. R.

CS Dep. Med., Univ. Kentucky, Lexington, KY, 40536, USA

SO Neuropharmacology (1990), 29(6), 579-84

CODEN: NEPHBW; ISSN: 0028-3908

DT Journal

LA English

AB The responses of resting plasma catecholamines, blood pressure, and heart rate were compared in rats receiving nicotine, administered either systematically or intracerebroventricularly (i.c.v.). Sympathoadrenal stress responses were also studied in rats rendered tolerant to nicotine from repeated systemic or intraventricular injections. Nicotine, given either intraventricularly or systematically, produced dose-related **increases** in the concn. of epinephrine in plasma. Little effect on norepinephrine in plasma was obsd. with nicotine given intraventricularly, indicating predominant stimulation of adrenomedullary pathways. In contrast, nicotine, given systematically, produced comparable **increases** in both epinephrine and norepinephrine. Blood pressure **increased** and heart rate fell in response to either intraventricular or systemic administration of nicotine. Rats exhibited tolerance to nicotine 24 h after a single intraventricular injection; however, tolerance was not detected with systematically injected nicotine unless the injections were given at least every 30 min. Whereas rats rendered tolerant to systemic administration of nicotine were cross-tolerant to stress, with respect to sympathoadrenal stimulation, cross-tolerance with stress was not detected in rats treated with nicotine repeatedly by the intraventricular route. Apparently, nicotinic receptors in brain modulate the central sympathetic outflow and adapt readily to nicotine stimulation with prolonged tolerance, but are probably not involved in sympathoadrenal stress responses. Peripheral nicotinic receptors, regulating sympathoadrenal secretion of catecholamines, displayed much shorter-lasting tolerance.

L7 ANSWER 35 OF 215 MEDLINE
 AN 94298160 MEDLINE
 DN 94298160 PubMed ID: 8026005
 TI Mechanisms responsible for sympathetic activation by cigarette **smoking** in humans.
 AU Grassi G; Seravalle G; Calhoun D A; Bolla G B; Giannattasio C; Marabini M; Del Bo A; Mancia G
 CS Cattedra Medicina Interna, Ospedale S. Gerardo dei Tintori, Monza, Italy.
 NC HL-02568 (NHLBI)
 HL-03220 (NHLBI)
 SO CIRCULATION, (1994 Jul) 90 (1) 248-53.
 Journal code: 0147763. ISSN: 0009-7322.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Abridged Index Medicus Journals; Priority Journals
 EM 199408
 ED Entered STN: 19940818
 Last Updated on STN: 19940818
 Entered Medline: 19940811
 AB BACKGROUND: The pressor and tachycardic effects of cigarette **smoking** are associated with an increase in plasma catecholamines, suggesting the dependence of these effects on adrenergic stimulation. Whether the stimulation occurs at a central or a peripheral level and whether reflex mechanisms are involved is unknown. METHODS AND RESULTS: In nine normotensive healthy subjects (age, 33.0 +/- 3.5 years, mean +/- SEM), we measured blood pressure (Finapres device), heart rate (ECG), calf blood flow and vascular resistance (venous occlusion plethysmography), plasma **norepinephrine** and **epinephrine** (high-performance liquid chromatography assay), and postganglionic muscle sympathetic nerve activity (microneurography from the peroneal nerve) while subjects were **smoking** a filter cigarette (**nicotine** content, 1.1 mg) or were in control condition. Cigarette **smoking** (which raised plasma **nicotine** measured by high-performance liquid chromatography from 1.0 +/- 0.9 to 44.2 +/- 7.1 ng/mL) markedly and significantly increased mean arterial pressure (+13.2 +/- 2.3%), heart rate (+30.3 +/- 4.7%), calf vascular resistance (+12.1 +/- 4.9%), plasma **norepinephrine** (+34.8 +/- 7.0%), and plasma **epinephrine** (+90.5 +/- 39.0%). In contrast, muscle sympathetic nerve activity showed a marked reduction (integrated activity -31.8 +/- 5.1%, $P < .01$). The reduction was inversely related to the increase in mean arterial pressure ($r = -.67$, $P < .05$), but the slope of the relation was markedly less (-54.1 +/- 7.5%, $P < .05$) than that obtained by intravenous infusion of phenylephrine in absence of **smoking**. The hemodynamic and neurohumoral changes were still visible 30 minutes after **smoking** and occurred again on **smoking** a second cigarette. Sham **smoking** was devoid of any hemodynamic and neurohumoral effect. CONCLUSIONS: These data support the hypothesis that in humans the sympathetic activation induced by **smoking** depends on an increased release and/or a reduced clearance of catecholamines at the neuroeffector junctions. Central sympathetic activity is inhibited by **smoking**, presumably via a baroreceptor stimulation triggered by the **smoking**-related pressor response. The baroreflex is impaired by **smoking**, however, indicating that partial inability to reflexly counteract the effect of sympathetic activation is also responsible for the pressor response.

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